STIC Search Report
Biotech-Chem Library

STIC Database Tracking Number: 121920

To: Sarvamangala Devi **Location:** REM 3C18 **Art Unit:** 1645 Monday, May 17, 2004 **Case Serial Number:** 10/030529	**From:** Beverly Shears **Location:** Remsen Bldg. RM 1A54 **Phone:** 571-272-2528 beverly.shears@uspto.gov

Search Notes

Shears, Beverly

From: Devi, Sarvamangala
Sent: Thursday, May 13, 2004 7:43 AM
To: Shears, Beverly
Subject: 10/030,529

Beverly:

Would please perform a sequence and an interference search for SEQ ID NO: 1 and SEQ ID NO: 2 and fragments thereof in application SN 10/030,529? Please also run the DNA sequence against the amino acid sequence.

Please include an invetors' name search: Christopher Elkins

Thanx.

S. DEVI, Ph.D.
AU 1645
Rems - 3C18

Devi, S.
10/030529


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      13may04 08:12:11 User219783 Session D2016.2
SYSTEM:OS  - DIALOG OneSearch
  File  65:Inside Conferences 1993-2004/May W2
        (c) 2004 BLDSC all rts. reserv.
  File 440:Current Contents Search(R) 1990-2004/May 13
        (c) 2004 Inst for Sci Info
  File 348:EUROPEAN PATENTS 1978-2004/May W01
        (c) 2004 European Patent Office
  File 357:Derwent Biotech Res. _1982-2004/May W2
        (c) 2004 Thomson Derwent & ISI
  File 113:European R&D Database 1997
        (c)1997 Reed-Elsevier(UK)Ltd All rts reserv
*File 113: This file is closed (no updates)

      Set  Items  Description
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Author

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Set     Items   Description
S1          8   AU=(ELKINS, C? OR ELKINS C?) AND (DSRA OR DSR(S)DUCREYI OR
             DUCREYI(5N)RESISTANCE)
S2          5   RD (unique items)
>>>No matching display code(s) found in file(s): 65, 113

 2/3,AB/1     (Item 1 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)
(c) 2004 Inst for Sci Info. All rts. reserv.

14916377  Document Delivery Available: 000178675100030  References: 44
TITLE: The Haemophilus ducreyi serum resistance antigen
    DsrA confers attachment to human keratinocytes
AUTHOR(S): Cole LE; Kawula TH (REPRINT); Toffer KL; Elkins C
AUTHOR(S) E-MAIL: kawula@med.unc.edu
CORPORATE SOURCE: Univ N Carolina, Dept Microbiol & Immunol, Campus Box
  7290/Chapel Hill//NC/27599 (REPRINT); Univ N Carolina, Dept Microbiol &
  Immunol, /Chapel Hill//NC/27599; Univ N Carolina, Dept Med, /Chapel
  Hill//NC/27599
PUBLICATION TYPE: JOURNAL
PUBLICATION: INFECTION AND IMMUNITY, 2002, V70, N11 (NOV), P6158-6165
GENUINE ARTICLE#: 605JQ
PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904
  USA
ISSN: 0019-9567
LANGUAGE: English   DOCUMENT TYPE: ARTICLE

ABSTRACT: Haemophilus ducreyi is the etiologic agent of the sexually
transmitted genital ulcer disease chancroid. H. ducreyi serum
resistance protein A (DsrA) is a member of a family of
multifunctional outer membrane proteins that are involved in resistance to
killing by human serum complement. The members of this family include YadA
of Yersinia species, the UspA proteins of Moraxella catarrhalis, and the
Eib proteins of Escherichia coli. The role of YadA, UspA1, and UspA2H as
eukaryotic cell adhesins and the function of UspA2 as a vitronectin binder
led to our investigation of the cell adhesion and vitronectin binding
properties of DsrA. We found that DsrA was a
keratinocyte-specific adhesin as it was necessary and sufficient for
attachment to HaCaT cells, a keratinocyte cell line, but was not required
for attachment to HS27 cells, a fibroblast cell line. We also found that
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DsrA was specifically responsible for the ability of H. ducreyi to bind vitronectin. We then theorized that **DsrA** might use vitronectin as a bridge to bind to human cells, but this hypothesis proved to be untrue as eliminating HaCaT cell binding of vitronectin with a monoclonal antibody specific to integrin alpha(v)beta(5) did not affect the attachment of H. ducreyi to HaCaT cells. Finally, we wanted to examine the importance of keratinocyte adhesion in chancroid pathogenesis so we tested the wild-type and **dsrA** mutant strains of H. ducreyi in our swine models of chancroid pathogenesis. The **dsrA** mutant was less virulent than the wild type in both the normal and immune cell-depleted swine models of chancroid infection.

2/3,AB/2 (Item 2 from file: 440)
DIALOG(R)File 440:Current Contents Search(R)

12441699 References: 41
TITLE: **DsrA**-deficient mutant of Haemophilus ducreyi is impaired in its ability to infect human volunteers
AUTHOR(S): Bong CTH; Throm RE; Fortney KR; Katz BP; Hood AF; **Elkins C**; Spinola SM (REPRINT)
AUTHOR(S) E-MAIL: sspinola@iupui.edu
CORPORATE SOURCE: Indiana Univ, Dept Med, 435 Emerson Hall,545 Barnhill Dr/Indianapolis//IN/46202 (REPRINT); Indiana Univ, Dept Med, /Indianapolis//IN/46202; Indiana Univ, Dept Microbiol & Immunol, /Indianapolis//IN/46202; Indiana Univ, Dept Pathol & Lab Med, /Indianapolis//IN/46202; Indiana Univ, Dept Dermatol, /Indianapolis//IN/46202; Univ N Carolina, Dept Med, /Chapel Hill//NC/27599; Univ N Carolina, Dept Microbiol & Immunol, /Chapel Hill//NC/27599
PUBLICATION TYPE: JOURNAL
PUBLICATION: INFECTION AND IMMUNITY, 2001, V69, N3 (MAR), P1488-1491
GENUINE ARTICLE#: 404GZ
PUBLISHER: AMER SOC MICROBIOLOGY, 1752 N ST NW, WASHINGTON, DC 20036-2904 USA
ISSN: 0019-9567
LANGUAGE: English DOCUMENT TYPE: ARTICLE

ABSTRACT: Haemophilus ducreyi produces an outer membrane protein called **DsrA**, which is required for serum resistance. An isogenic **dsrA** mutant, FX517, was constructed previously in H. ducreyi 35000, Compared to its parent, FX517 cannot survive in normal human serum. When complemented in trans with a plasmid containing **dsrA**, FX517 is converted to a serum-resistant phenotype (C, Elkins, K, J, Morrow, Jr,, and B, Olsen, Infect. Immun, 68:1608-1619, 2000), To test whether **dsrA** was transcribed in vivo, we successfully amplified transcripts in five biopsies obtained from four experimentally infected human subjects. To test whether **DsrA** was required for virulence, six volunteers were experimentally infected,vith 35000 and FX517 and observed for papule and pustule formation. Each subject was inoculated with two doses (70 to 80 CFU) of live 35000 and 1 dose of heat-killed bacteria on one arm and with three doses (ranging from 35 to 800 CFU) of live FX517 on the other arm. Papules developed at similar rates at sites inoculated with the mutant or parent. However, mutant papule surface areas were significantly smaller than parent papules. The pustule formation rate was 58% (95% confidence interval [CI]

of 28 to 85%) at 12 parent sites, and 0% (95% CI of 0 to 15%) at 18 mutant sites (P = 0.0004), Although biosafety regulations precluded our testing the complemented mutant in humans, these results suggest that expression of **DsrA** facilitates the ability of H. ducreyi to progress to the pustular stage of disease.

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13may04 08:19:52 User219783 Session D2016.3